Neuroimaging Support for Discrete Neural Correlates of Basic Emotions: A Voxel-based Meta-analysis

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Abstract

■ What is the basic structure of emotional experience and how is it represented in the human brain? One highly influential theory, discrete basic emotions, proposes a limited set of basic emotions such as happiness and fear, which are characterized by unique physiological and neural profiles. Although many studies using diverse methods have linked particular brain structures with specific basic emotions, evidence from individual neuroimaging studies and from neuroimaging meta-analyses has been inconclusive regarding whether basic emotions are associated with both consistent and discriminable regional brain activations. We revisited this question, using activation likelihood estimation (ALE), which allows spatially sensitive, voxel-wise statistical comparison of results from multiple studies. In addition, we examined substantially more studies than previous meta-analyses. The ALE meta-analysis yielded results consistent with basic emotion theory. Each of the emotions examined (fear, anger, disgust, sadness, and happiness) was characterized by consistent neural correlates across studies, as defined by reliable correlations with regional brain activations. In addition, the activation patterns associated with each emotion were discrete (discriminable from the other emotions in pairwise contrasts) and overlapped substantially with structure–function correspondences identified using other approaches, providing converging evidence that discrete basic emotions have consistent and discriminable neural correlates. Complementing prior studies that have demonstrated neural correlates for the affective dimensions of arousal and valence, the current meta-analysis results indicate that the key elements of basic emotion views are reflected in neural correlates identified by neuroimaging studies. ■

INTRODUCTION

Emotions are a key facet of human experience. A central question in the study of emotion is how best to characterize the basic structure of emotional experience. Discrete emotion theories (Ekman, 1972; Darwin, 1872) propose a limited set of basic emotions (e.g., happiness, sadness, anger, fear, and disgust) that have unique physiological and neural profiles. Other theoretical views, such as dimensional theories of emotion, conceptualize emotions using a framework in which affective states can be represented in terms of underlying factors such as emotional arousal (emotion strength) and emotional valence (degree of pleasantness or unpleasantness).

A key proposal of basic emotion theories is that basic emotions have consistent and specific psychophysiological and neural correlates. Ekman (1999) summarized this view: "It is necessary to posit emotion-specific central nervous system (CNS) activity in my account of basic emotions. The distinctive features of each emotion, including the changes not just in expression but in memories, imagery, expectations and other cognitive activities, could not occur without central nervous system organization and direction. There must be *unique* physiological [CNS] patterns for each emotion (p. 50)". Although the predictions of basic emotion theories have drawn support from a wide variety of behavioral, neuropsychological, psychophysiological, and neuroimaging studies (e.g., Damasio et al., 2000; Blair, Morris, Frith, Perrett, & Dolan, 1999; Ekman, 1992), recently the strength of the support for basic emotion theories has been challenged (e.g., Barrett, Lindquist, Bliss-Moreau, Duncan, & Brennan, 2007; Barrett & Wager, 2006; Barrett & Russell, 1999). For example, reviews of the psychophysiological literature have concluded that such studies have not been able to identify consistent and specific psychophysiological correlates for basic emotions (Barrett & Wager, 2006; Cacioppo, Berntson, Larsen, Poehlmann, & Ito, 2000; Zajonc & McIntosh, 1992).

Neuroimaging studies can assess activity related to emotional states across the entire brain on a moment-to-moment basis, and thus one might expect that this approach would be more sensitive and better able to identify the consistent and specific biological correlates for basic emotions than other measures such as behavior or psychophysiology. However, the strength and the consistency of the neuroimaging evidence supporting the predictions of basic emotion theories have also been questioned, and some critiques have concluded that evidence for basic emotions from neuroimaging remains inconclusive (Barrett & Wager, 2006; Barrett & Russell, 1999). The existing literature directly relevant to evaluating whether basic emotions have differentiable neural correlates is relatively limited, in

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part because only a handful of neuroimaging studies have examined and contrasted several basic emotions concurrently in the same study. Meta-analytic methods applied to the neuroimaging literature can help overcome this limitation in the available corpus of literature because such methods allow activation patterns to be compared across different studies. Such techniques can identify neural patterns that are consistent and specific to each emotion state. Meta-analyses can also assess whether these activation patterns are robust across experimental differences such as type of emotional stimuli and emotion-elicitation methods, and they can reduce problems associated with low experimental power in individual studies (Ioannidis & Lau, 1999).

Two meta-analytic reviews of the relevant basic emotion neuroimaging literature have been conducted to date (Murphy, Nimmo-Smith, & Lawrence, 2003; Phan, Wager, Taylor, & Liberzon, 2002; for additional meta-analytic reviews of the neural correlates of emotion, but not basic emotion states, see also Kober et al., 2008; Baas, Aleman, & Kahn, 2004; Wager, Phan, Liberzon, & Taylor, 2003). Both Phan et al. (2002) and Murphy et al. (2003) concluded that basic emotion theories are only partially supported by neuroimaging studies, and each review reached somewhat different conclusions regarding which specific neural correlates are associated with each basic emotion (Barrett & Wager, 2006). Because the status of the neuroimaging evidence supporting basic emotion theories is currently unresolved, we revisited these questions in the current meta-analytic study. We hypothesized that by using a more sensitive meta-analytic method (activation likelihood estimation; ALE, Laird et al., 2005) than those used in previous reviews and by analyzing a substantially larger number of neuroimaging studies that have been published in the several years following the publication of these earlier reviews, we could potentially reveal differences between basic emotion states that were not detected in previous studies.

The current study differs from previous meta-analytic reviews in two primary respects: the meta-analytic methodology used and the number of studies included. We used the ALE method, which preserves three-dimensional spatial information in the original activation maximum coordinate data, unlike label-based methods that convert activation coordinates into regional labels (e.g., pFC), decreasing spatial information considerably. ALE allows for direct statistical comparison between the composite activation maps associated with discrete emotion states and thus provides a means for assessing the discriminability of basic emotion states at the voxel level. Although the analysis used by Murphy et al. (2003) did assess the differentiability of neural patterns associated with basic emotions states, their meta-analysis method divided the brain into only eight sectors of approximately equal volume. These sectors are larger than individual brain structures and are orders of magnitude less spatially specific than the voxel level resolution afforded by ALE. Thus, this prior study could not assess the critical question relevant to the predictions of basic emotion theory, namely, whether basic emotions have consistent and specific correlates at the level of individual brain structures. Similarly, Phan et al. (2002) did not specifically assess whether each basic emotion could be discriminated from each of the other emotions on the basis of regional activations. Their meta-analysis focused on determining which particular brain regions were more consistently associated with one particular emotion than other emotions, and it did not assess the discriminability of basic emotions at any level. In addition to the methodological advantages associated with the current ALE meta-analysis, our review examined the considerably enlarged literature (50% more studies published subsequent to the most recent meta-analytic review; Murphy et al., 2003) that has resulted from the recent increase in the number of neuroimaging studies examining the neural correlates of emotion. Although the majority of studies have explored the neural correlates of basic emotions using facial emotion stimuli, more recent studies have increasingly adopted a broader range of stimuli and methods. Together, these two considerations motivated a reexamination of whether the existing neuroimaging evidence supports the basic emotion view.

To address whether there are differentiable patterns of neural activity specific to each basic emotion we conducted two primary types of analysis, which can be characterized as assessing the *consistency* and *discriminability* of emotion-related activations, respectively. Consistency analyses determined the brain regions whose activity was most consistently and strongly associated with each of the individual basic emotions. Basic emotion theories predict that there should be characteristic regional brain activations that are reliably associated with the experience of each basic emotion. These neural correlates are also predicted to be discrete or discriminable, in the sense that each basic emotion is associated with some unique regional activations not shared by the other emotions. To test this prediction, we contrasted the activations associated with each basic emotion, assessing whether patterns of regional brain activation can discriminate between different basic emotions. The degree of support or lack of support for basic emotion theories was assessed primarily on the extent to which basic emotions were associated with consistent and discriminable regional activations.

In addition, we anticipated that the regions identified in the consistency and discriminability analyses would overlap to some degree on the basis of the view that some subset of the characteristic neural activations for each emotion also would comprise the activations that differentiated that emotion from others. Finally, we also predicted that the characteristic patterns of regional brain activity associated with basic emotions as observed with neuroimaging should converge with the regions identified using other neuroscience methods such as neuropsychological studies. For example, because neuropsychological lesion studies in humans have demonstrated that the amygdala

is a structure critically implicated in the experience of fear and the acquisition of fear responses, one would predict that the amygdala should be among the brain regions characteristic of the basic emotion fear in our meta-analysis (Adolphs, Tranel, Damasio, & Damasio, 1994).

METHODS

Scope of the Review

To investigate patterns of neural activation associated with discrete basic emotions, we examined neuroimaging studies that included either an explicit emotional elicitation task (e.g., mood induction), emotionally arousing stimuli (e.g., emotional pictures), or emotional facial expressions. Like Murphy et al. (2003), the current analysis considered studies that addressed any aspect of an emotional experience: expression, perception, interpretation, or subjective experience. Consequently, our meta-analysis examined neural activations across multiple studies that recruited a variety of different emotion-related processes. We elected to include all such studies rather than focus on studies using a particular methodology such as emotion induction because we were specifically interested in identifying the "core" neural patterns associated with basic emotions, reflected in the overlap of activations across different aspects of emotional experience.

Studies were selected based on a set of seven criteria that were adapted from inclusion criteria used in previous meta-analyses (e.g., Murphy et al., 2003; Phan et al., 2002). First, only studies conducted using $H_2{}^{15}O$ PET and fMRI were considered. Second, coordinates needed to be reported in standard stereotactic space (either MNI or Talairach). Third, studies must have reported whole-brain analyses (we excluded those studies reporting only ROI analyses) to ensure that all regions in the brain were represented equivalently. Fourth, activation contrasts representing main effects of specific emotions relative to a baseline condition were required (e.g., viewing happy faces > viewing neutral faces) so that the activations associated with each emotion could be analyzed independently of any other emotion. This criterion also reduced the influence of stimulus type on the reported effects because effective control stimuli were well matched on all elements except for emotional arousal. Fifth, the main effects reported in a study were required to include at least one basic emotion state (happiness, sadness, anger, fear, or disgust). Sixth, studies had to report activations (deactivations were not included in the analysis because the nature of the analysis technique did not afford differentiation of activations from deactivations). Seventh, only data from healthy individuals were included (studies of clinical patient groups were not considered).

Over 1,000 potential studies were identified by a search of electronic databases (PsychInfo, Medline, Web of Science ISI), Google Scholar, previous meta-analyses (Murphy et al., 2003; Phan et al., 2002), and relevant peer-reviewed journals. Eighty-three neuroimaging studies (PET and fMRI) published from 1993 to 2008 were selected for the analysis (for a summary, see Table 1). The current analysis included 30 studies (approximately 100% more than Phan et al., 2002 and 50% more than Murphy et al., 2003) published after the studies included in the most recent meta-analysis (Murphy et al., 2003). Studies included in the ALE meta-analysis are preceded in the References section by an asterisk.

Activation Likelihood Estimation

The current review used a recently developed neuroimaging meta-analysis method, ALE (Laird et al., 2005), which has considerable advantages over previously used label-based methods where anatomic locations of activations are analyzed according to their corresponding neural structures. ALE is a quantitative method of assessing relationships between function (i.e., cognitive or emotional processes) and regional brain activations. In an ALE analysis, relevant neuroimaging studies are collected and analyzed in relation to specific experimental conditions (e.g., viewing a frightening scene vs. a neutral scene). Three-dimensional focus of activation is extracted in the form of Talairach or MNI coordinates corresponding to activation maxima for contrasts between experimental conditions. These sets of activation coordinates are then modeled as the centers of Gaussian probability distributions and are combined (summated) to create statistical whole-brain ALE maps. ALE maps preserve considerably more spatial information from the original maxima, relative to label-based methods, and substantially increase the spatial sensitivity of the analysis. The ALE maps are comprised of ALE statistics representing the likelihood that the voxel at that three-dimensional coordinate is active during the corresponding experimental condition across the entire set of studies analyzed (Laird et al., 2005). A further advantage of the ALE method is that these individual ALE maps can then be directly compared statistically, by contrasting the voxelwise differences between two ALE maps and comparing the resulting difference ALE map to a comparison null distribution generated by random permutation tests. To summarize the steps in the current ALE meta-analysis (for a complete description of the ALE method, see Laird et al., 2005), three-dimensional activation coordinates were extracted from the relevant studies for each basic emotion, converted to spatially smoothed activation foci volumes with a 10-mm FWHM Gaussian kernel, and pooled across studies to create statistical whole-brain maps using GingerALE 1.1 (Laird et al., 2005).

For consistency analyses, ALE statistic maps were calculated for each of the five basic emotions analyzed, and each ALE map was then compared with a corresponding comparison null distribution of the ALE statistic based on 5,000 random spatial permutations across the brain of an equivalent number of activation foci. Similarly, for discriminability analyses, ALE statistic maps were compared by

Table 1. Studies Included in the Meta-analysis

Study	*Method*	*n*	*Age*	*Experimental Paradigm*	*Modality*	*Emotion*
Aalto et al. (2002)	PET	11f	18–44	Mood induction	V (Films)	S
Aalto et al. (2005)	fMRI	11f	33.4	Viewing emotional films	V (Films)	S
Abel et al. (2003)	fMRI	8m	N/A	Viewing facial expressions	V (Faces)	F
Abler, Erk, Herwig, and Walter (2007)	fMRI	12f	40.7	Viewing emotional pictures	V (Pictures)	D
Ashwin, Baron-Cohen, Wheelwright, O'Riordan, and Bullmore (2007)	fMRI	13m	25.6	Viewing facial expressions	V (Faces)	F
Baker, Frith, and Dolan (1997)	fMRI	10m	18–35	Mood induction	V (Scripts/Music)	H S
Beauregard et al. (1998)	fMRI	3m, 4f	45	Viewing emotional films	V (Films)	S
Benuzzi et al. (2004)	fMRI	7m, 7f	21–27	Viewing facial expressions	V (Faces)	F
Benuzzi, Lui, Duzzi, Nichelli, and Porro (2008)	fMRI	15f	23.5	Viewing emotional films	V (Films)	D
Blair et al. (1999)	PET	13m	25	Viewing facial expressions	V (Faces)	A
Buchanan et al. (2000)	fMRI	10m	22–40	Emotional prosody	A (Voices)	H S
Bystritsky et al. (2001)	fMRI	3m, 3f	31.8	Mood induction	A (Autobio Scripts)	F
Damasio et al. (2000)	PET	53mix	N/A	Induced mood	Autobio Recall	H S A F
Dolan et al. (1996)	PET	8m	23	Viewing facial emotions	V (Faces)	H
Dougherty et al. (1999)	PET	8m	25	Mood induction	A (Autobio Scripts)	A
Eugene et al. (2003)	fMRI	10f	24	Viewing emotional films	V (Films)	S
Fischer et al. (2005)	fMRI	11m, 11f	74.1	Viewing facial expressions	V (Faces)	A
Fitzgerald et al. (2004)	fMRI	7m, 5f	31.2	Mood induction	Autobio Recall	D
Fitzgerald, Angstadt, Jelsone, Nathan, and Phan (2006)	fMRI	10m, 10f	26	Viewing facial expressions	V (Faces)	H S A F D
George et al. (1995)	PET	11f	N/A	Induced mood	Autobio Recall/V (Faces)	S
George, Ketter, Parekh, Herscovitch, and Post (1996)	PET	10m, 10f	35	Induced mood	Autobio Recall/V (Faces)	H S
Goldin et al. (2005)	fMRI	13f	19.7	Viewing emotional films	V (Films)	H S
Grandjean et al. (2005)	fMRI	8m, 7f	24.4	Emotional prosody	A (Pseudo Sentences)	A
Grosbras and Paus (2005)	fMRI	10m, 10f	28.6	Viewing emotional films	V (Films)	A
Habel, Klein, Kellermann, Shah, and Schneider (2005)	fMRI	26m	33.4	Mood induction	V (Faces)	H S
Hadjikhani et al. (2003)	fMRI	4m, 3f	N/A	Viewing bodily expressions	V (Bodily Expressions)	F
Hariri, Mattay, Tessitore, Fera, and Weinberger (2003)	fMRI	5m, 6f	32	Viewing emotional pictures	V (Pictures)	F
Harris and Fiske (2007)	fMRI	10mix	N/A	Viewing emotional pictures	V (Pictures)	D
Hutcherson et al. (2005)	fMRI	28f	18–21	Viewing emotional films	V (Films)	H S
Kesler/West et al. (2001)	fMRI	11m, 10f	21.6	Processing facial emotions	V (Faces)	H S A F

Table 1. (*continued*)

Study	*Method*	*n*	*Age*	*Experimental Paradigm*	*Modality*	*Emotion*
Killgore and Yurgelun-Todd (2004)	fMRI	12f	23.7	Viewing facial expressions	V (Faces)	H S
Kilts, Egan, Gideon, Ely, and Hoffman (2003)	fMRI	9m, 4f	24.5	Viewing facial expressions	V (Faces)	H A
Kimbrell et al. (1999)	PET	10m, 8f	31.2, 34.7	Induced mood	Autobio Recall	F
Lane, Reiman, Ahern, Schwartz, and Davidson (1997)	PET	12f	23.3	Induced mood	V (Film)/Recall	H S D
Lange et al. (2003)	fMRI	9m	29	Viewing facial expressions	V (Faces)	F
Lemche et al. (2007)	fMRI	5f, 7m	27.3	Viewing facial expressions	V (Faces)	H S
Lennox, Jacob, Calder, Lupson, and Bullmore (2004)	fMRI	6m, 6f	32.6	Viewing facial expressions	V (Faces)	H S
Liddell et al. (2005)	fMRI	11m, 11f	32	Viewing facial expressions	V (Faces)	F
Liotti et al. (2000)	PET	8f	N/A	Mood induction	V (Autobio Scripts)	S
Mayberg et al. (1999)	PET	8f	36	Mood induction	V (Autobio Scripts)	S
Michalopoulou et al. (2008)	fMRI	5m, 4f	32	Viewing facial expressions	V (Faces)	F
Mitterschiffthaler, Fu, Dalton, Andrew, and Williams (2007)	fMRI	8m, 8f	30.8	Mood induction	A (Music)	H S
Moll et al. (2005)	fMRI	7m, 6f	22.5	Mood induction	V (Statements)	D
Morris et al. (1998)	PET	4m, 1f	42.8	Viewing facial expressions	V (Faces)	H F
Ottowitz et al. (2004)	fMRI	8f	18–30	Mood induction	V (Sentences)	S
Paradiso et al. (1997)	PET	2m, 6f	62.6	Viewing emotional films	V (Film Clips)	H D
Paradiso, Robinson, Boles Ponto, Watkins, and Hichwa (2003)	fMRI	9m, 8f	65	Mood induction	V (Faces/Pictures)	S
Pardo, Pardo, and Raichle (1993)	PET	3f	24	Mood induction	Imagery	S
Pelletier et al. (2003)	fMRI	5m, 4f	33	Mood induction	V (Autobio Recall)	H S
Phillips et al. (1997)	fMRI	2m, 5f	27	Viewing facial expressions	V (Faces)	F D
Phillips, Bullmore, et al. (1998)	fMRI	7m, 1f	32	Viewing facial expressions	V (Faces)	H S
Phillips, Young, et al. (1998)	fMRI	6m	37	Vocal expressions	V (Faces)/A (Vocal)	F D
Phillips et al. (1999)	fMRI	5mix	30	Viewing facial expressions	V (Faces)	A F D
Phillips et al. (2000)	fMRI	7m, 7f	31	Viewing emotional pictures	V (Pictures)	D
Phillips et al. (2004)	fMRI	5m, 5f	29.5	Viewing facial expressions	V (Faces)	F D
Pietrini, Guazzelli, Basso, Jaffe, and Grafman (2000)	PET	8m, 7f	22	Mood induction	Imagery	A
Pine et al. (2001)	fMRI	10m, 10f	13.9, 28.5	Visual masking paradigm	V (Faces)	H F
Salloum et al. (2007)	fMRI	11m	36	Viewing facial expressions	V (Faces)	H S A F D
Sambataro et al. (2006)	fMRI	11m, 13f	26.8	Viewing facial expressions	V (Faces)	D

Table 1. (*continued*)

Study	*Method*	*n*	*Age*	*Experimental Paradigm*	*Modality*	*Emotion*
Sato, Kochiyama, Yoshikawa, Naito, and Matsamura (2004)	fMRI	10m, 12f	26.5	Viewing facial expressions	V (Dynamic Faces)	H F
Schafer, Schienle, and Vaitl (2005)	fMRI	20m, 20f	23.93	Viewing emotional pictures	V (Pictures)	F
Schienle et al. (2002)	fMRI	12f	26.3	Viewing emotional pictures	V (Pictures)	F D
Schienle, Schäfer, Walter, Stark, and Vaitl (2005)	fMRI	63f	27.3	Viewing emotional pictures	V (Pictures)	D
Schienle et al. (2006)	fMRI	12f	19–41	Viewing emotional pictures	V (Pictures)	F D
Shapira et al. (2003)	fMRI	3m, 5f	38	Viewing emotional pictures	V (Pictures)	D
Sprengelmeyer, Rausch, Eysel, and Przuntek (1998)	fMRI	2m, 4f	23.5	Recognition of facial expressions	V (Faces)	A F D
Stark et al. (2003)	fMRI	4m, 11f	29.1	Viewing emotional films	V (Pictures)	F D
Stark et al. (2005)	fMRI	6m	N/A	Viewing emotional pictures	V (Films)	F D
Stark et al. (2007)	fMRI	34m, 32f	24.7	Viewing emotional pictures	V (Pictures)	F D
Takahashi et al. (2008)	fMRI	8m, 8f	21.5	Mood induction	V (Sentences)	H
Thielscher and Pessoa (2007)	fMRI	10m, 15f	23	Viewing facial expressions	V (Faces)	F D
Vuilleumier and Pourtois (2007)	fMRI	12mix	N/A	Viewing facial expressions	V (Faces)	F
Wang, McCarthy, Song, and LaBar (2005)	fMRI	5m, 7f	25.9	Visual oddball task	V (Pictures)	S
Whalen et al. (1998)	fMRI	4m, 4f	25	Viewing facial expressions	V (Faces)	A F
Wicker et al. (2003)	fMRI	14m	N/A	Mood induction	O	D
Williams et al. (2001)	fMRI	11m	30	Viewing facial expressions	V (Faces)	A
Williams et al. (2004)	fMRI	15m, 7f	27.5	Viewing facial expressions	V (Faces)	F
Williams et al. (2005)	fMRI	5m, 8f	24	Viewing facial expressions	V (Faces)	A F D
Winston, Vuilleumier, and Dolan (2003)	fMRI	6m, 8f	30	Viewing facial expressions	V (Faces)	F
Wright, He, Shapira, Goodman, and Liu (2004)	fMRI	4m, 4f	20–26	Viewing emotional pictures	V (Pictures)	F D

Characteristics of all studies included in the meta-analysis. Abbreviations for stimulus modality: V = visual; A = auditory; O = olfactory; for emotion category: H = happiness; S = sadness; A = anger; F = fear; D = disgust; experimental paradigm: Autobio = autobiographical.

contrasting the difference maps calculated from each pairwise contrast between individual emotion ALE maps (e.g., fear ALE map minus anger ALE map) across all basic emotions with a corresponding random null distribution. This null distribution was calculated, first, by generating 5,000 individual pairs of ALE maps, using the same permutation method as was used to compute individual ALE maps; second, by calculating a difference map for each pair; and third, by comparing the observed difference ALE map between the emotion pair with this null distribution. All thresholded ALE maps were corrected for multiple comparisons using the false discovery rate algorithm ($q = .05$) and were overlaid on a canonical single-subject anatomical T1 brain template from the SPM5 image library. Only significant clusters that exceeded 100 mm^3 were reported.

In summary, the ALE meta-analysis was comprised of consistency analyses and discriminability analyses. Consistency analyses identified the regional brain activations regions most consistently associated with each basic emotion. Discriminability analyses identified brain regions that were significantly differentially active when contrasting pairs of discrete emotions, thus addressing whether

basic emotion states are discriminable based on regional activations.

RESULTS

Activation Consistency Analyses

Happiness

The ALE analysis of activation foci associated with happiness revealed nine significant clusters, with the largest (4880 mm^3) located primarily in the right superior temporal gyrus (STG; Brodmann's area [BA] 22; see Figure 1 and Table 2). Figure 1 displays ALE activation maps overlaid on eight axial slices from a canonical T1 anatomical image, centered on $z = 0$, with the highest slice selected at a level that captured the most superior activation(s) across all statistical maps in the meta-analysis. The same display criteria were applied to all figures.

Sadness

The ALE analysis of activation foci associated with sadness revealed 35 significant clusters, with the largest (3120 mm^3) located primarily in the left medial frontal gyrus (medFG; see Figure 1 and Table 2).

Anger

The ALE analysis of activation foci associated with anger revealed 13 significant clusters, with the largest (2408 mm^3)

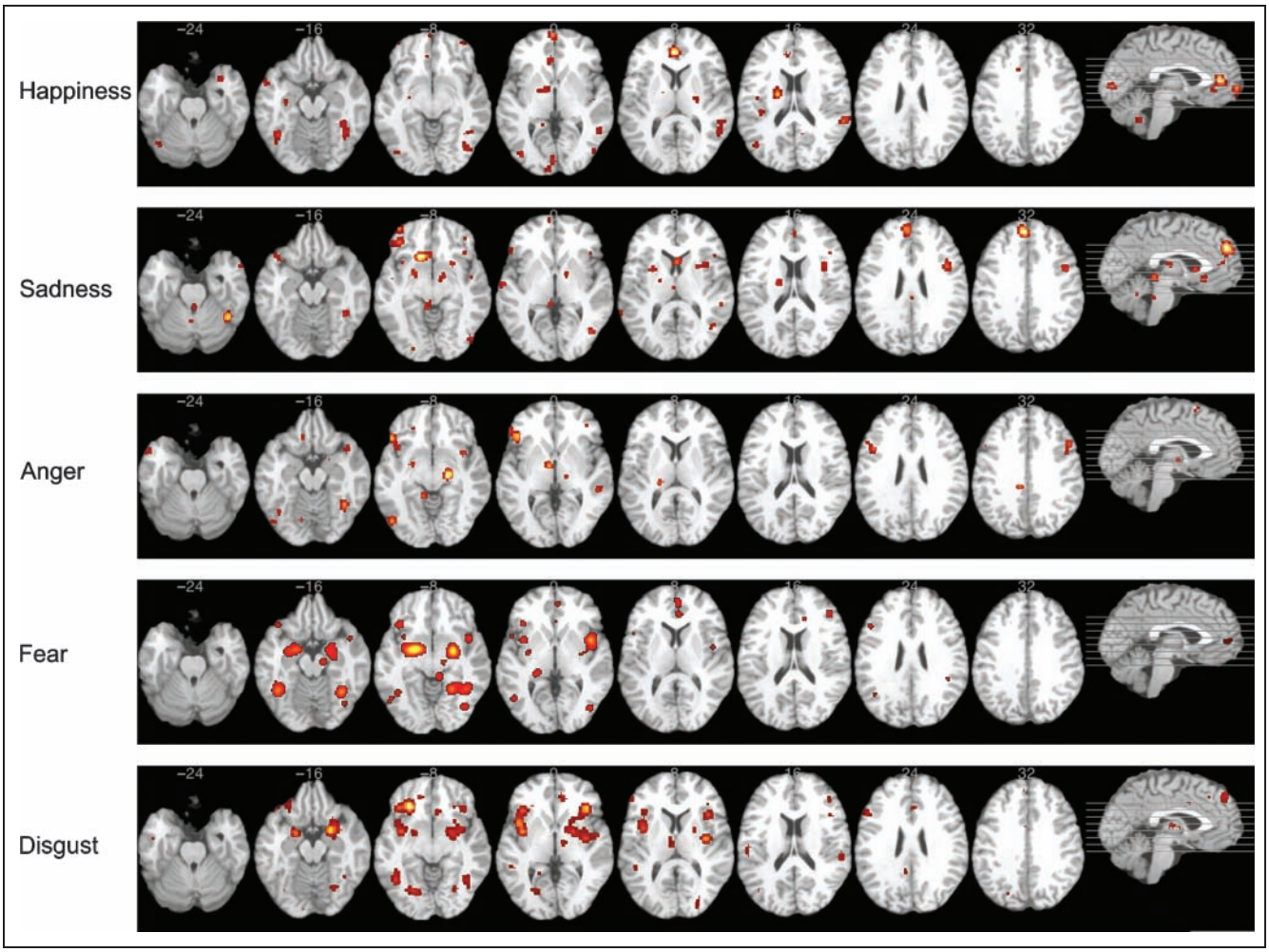


Figure 1. Activation likelihood maps representing regional activity consistently associated with each basic emotion state. Statistical map of significant ALE clusters associated with happiness, sadness, anger, fear, and disgust. The horizontal lines overlaid on the sagittal image (at far right) show the locations of the corresponding axial slices. All figures display slices in neurological convention, where the left side in the image corresponds to the left side of the image. ALE values are indicated by red-yellow color gradient clusters overlaid on a canonical structural image from SPM5. Rather than representing magnitude of activation, the color gradient represents the degree of overlap (i.e., activation likelihood or consistency) among the activation coordinates across studies that contributed to the analysis. The most prominent clusters associated with happiness are located in right STG (BA 22) and left ACC (BA 24). The most prominent clusters associated with sadness are located in left caudate head and left medFG (BA 9) and right IFG (BA 9). The most prominent clusters associated with anger are located in left IFG (BA 47) and right parahippocampal gyrus (BA 35). The most prominent clusters associated with fear are located in bilateral amygdala, right cerebellum, and right insula. The most prominent clusters associated with disgust are located in bilateral insula (BA 47).

Table 2. ALE Activation Clusters Consistently Associated with Each Basic Emotion State

Activation Focus				
x	*y*	*z*	*Region (>100 mm³)*	*Size*
Happiness				
47.8	−52.9	−0.6	R STG (BA 22)[a]	4880
−1.9	42.3	4.2	L ACC (BA 24)[a]	3232
−40.2	−61.9	−18.4	L cerebellum[a]	1176
−18.3	−9.3	16.6	L thalamus	960
−4.2	−91.5	1.7	L lingual gyrus	888
−12.2	−5.7	1.5	L thalamus	824
−39.1	−78.8	−2.8	L Inf Occ gyrus[a]	528
−36.9	−31.0	18.0	L insula[a]	288
24.7	−16.0	7.8	R basal ganglia (Put)[a]	200
Anger				
−44.4	22.5	−3.4	L IFG (BA 47)[a]	2408
18.6	−19.4	−8.1	R PHG	1544
−43.6	−70.7	−11.3	L fusiform gyrus[a]	1480
39.1	8.0	−14.5	R IFG (BA 13)	1008
37.3	−54.5	−15.7	R cerebellum[a]	1000
48.1	13.1	30.0	R MFG (BA 9)[a]	928
−45.2	11.5	25.9	L IFG (BA 9)[a]	904
−6.1	−8.5	1.0	L thalamus[a]	568
−50.8	7.7	−22.4	L STG	464
−22.7	−7.3	−8.0	L amygdala	128
4.6	45.0	−4.0	R ACC (BA 32)[a]	128
−11.0	24.0	−16.2	L medFG (BA 25)	120
12.0	−23.0	64.0	R medFG (BA 6)[a]	112
Fear				
−22.7	−5.9	−9.0	L amygdala[a]	5616
22.7	−10.6	−11.1	R amygdala[a]	4248
32.6	−53.3	−9.9	R cerebellum[a]	4176
42.7	2.7	−1.5	R insula (BA 13)	2896
−40.1	−55.7	−13.8	L fusiform gyrus[a]	2848
−37.5	22.6	−7.4	L IFG (BA 47)[a]	1320
4.0	43.6	4.8	R ACC (BA 32)	1168
38.6	−73.0	−7.4	R Inf Occ gyrus[a]	1072
37.7	10.4	19.9	R insula (BA 13)[a]	368
42.5	−40.2	20.6	R Insula (BA 13)[a]	320
13.0	29.7	13.7	R ACC (BA 32)[a]	176
Sadness				
−3.5	46.8	27.1	L medFG (BA 9)[a]	3120
39.3	6.4	20.9	R IFG (BA 9)	2576
−9.8	17.7	−8.3	L caudate head[a]	1960
−38.3	39.9	−7.6	L MFG (BA 10)[a]	1632
40.0	−51.1	−21.5	R cerebellum[a]	1344
43.4	−66.3	4.2	R ITG	880
−4.6	−38.8	−5.2	L cerebellum[a]	840
1.8	11.6	6.2	R caudate head	816
−16.5	−11.6	13.9	L thalamus[a]	808
13.0	−5.3	−6.2	R PHG[a]	784
−36.5	13.6	−13.5	L IFG (BA 13)	632
2.9	7.8	62.0	R SFG[a]	512
−47.2	−6.6	41.1	L precentral gyrus	496
44.4	−78.4	−10.4	R middle Occ gyrus	456
−20.4	−1.2	−7.4	L basal ganglia (GP)	408
−59.2	−14.7	−0.7	L STG	400
39.6	21.5	−4.2	L IFG (BA 47)	352
−26.3	2.7	9.1	L basal ganglia (Put)	336
44.2	21.1	12.2	R IFG (BA 45)	272
23.5	8.6	−6.9	R basal ganglia (Put)	208
−49.9	25.2	0.5	L IFG (BA 45)	208
33.1	−21.9	19.2	R insula (BA 13)	128
Disgust				
30.4	4.4	−3.5	R IFG (BA 47/Insula)[a]	14208
−26	28	−10	L IFG (BA 47/Insula)[a]	10720
−22.0	−70.0	−6.0	L lingual gyrus[a]	1800
−19.7	−3.3	−13.8	L amygdala	1352
−41.0	−55.2	−9.0	L fusiform gyrus[a]	1272
39.8	−58.2	−9.2	R fusiform gyrus	1104
−1.6	43.6	39.7	L medFG	960
26.7	−67.3	−12.3	R cerebellum[a]	680
−49.7	18.8	26.3	R IFG (BA 9)	672
−4.3	−13.9	7.1	L thalamus	512
−47.4	−43.6	3.9	L MTG	472
26.7	−83.1	9.7	R middle Occ gyrus	408
9.6	37.6	−0.6	R ACC	384
6.9	20.7	−8.7	R ACC (BA 32)	288
−13.6	38.2	−6.9	L medFG (BA 10)	264
−49.7	36.1	9.0	L IFG (BA 46)	200

Each cluster greater than 400 mm³ is reported, along with the weighted central activation likelihood focus, the region corresponding to the highest ALE score within the cluster, and the total cluster size in mm³. Additional clusters of interest that surpassed a threshold of 100 mm³ are also reported. L and R indicate activations located in the left and right hemispheres, respectively. Inf = inferior; Occ = occipital; GP = globus pallidus; Put = putamen; PHG = parahippocampal gyrus. BAs are provided to differentiate activations in larger regions that occur in multiple contrasts.

[a]Indicates regions overlapping with the reanalysis that involved only studies that used facial expressions.

located primarily in the left inferior frontal gyrus (IFG; BA 47; see Figure 1 and Table 2).

Fear

The ALE analysis of activation foci associated with fear revealed 11 significant clusters, with the largest (5616 mm^3) located primarily in the left amygdala (see Figure 1 and Table 2).

Disgust

The ALE analysis of activation foci associated with disgust revealed 16 significant clusters, with the largest (14208 mm^3) located primarily in the right insula and right IFG (BA 47; see Figure 1 and Table 2).

Activation Discriminability Analyses

Happiness–Sadness

The ALE analysis of activation foci associated with happiness greater than sadness revealed four significant clusters, with the largest (424 mm^3) located primarily in the right STG (see Figure 2 and Table 3). The ALE analysis of activation foci associated with sadness greater than happiness revealed 12 significant clusters, with the largest (2536 mm^3) located primarily in the right middle temporal gyrus (MTG; BA 24; see Figure 2 and Table 3). For all contrast analysis figures, clusters displayed in the red gradient correspond to the emotion state that is being subtracted from in the contrast; clusters displayed in the blue gradient correspond to the emotion state that is being subtracted.

Happiness–Anger

The ALE analysis of activation foci associated with happiness greater than anger revealed six significant clusters, with the largest (1032 mm^3) located primarily in the left rostral ACC (BA 32; see Figure 2 and Table 3). The ALE analysis of activation foci associated with anger greater than happiness revealed six significant clusters, with the largest (1536 mm^3) located primarily in the IFG (BA 47; see Figure 2 and Table 3).

Happiness–Fear

The ALE analysis of activation foci associated with happiness greater than fear revealed six significant clusters, with the largest (1592 m^3) located primarily in the right STG (BA 22; see Figure 2 and Table 3). The ALE analysis of activation foci associated with fear greater than happiness revealed 11 significant clusters, with the largest (3192 m^3) located primarily in the left amygdala.

Happiness–Disgust

The ALE analysis of activation foci associated with happiness greater than disgust revealed four significant clusters, with the largest (672 mm^3) located primarily in the left rostral ACC (BA 24; see Figure 2 and Table 3). The ALE analysis of activation foci associated with disgust versus happiness revealed 11 significant clusters, with the largest (12008 mm^3) located primarily in the right putamen (see Figure 2 and Table 3).

Sadness–Anger

The ALE analysis of activation foci associated with sadness greater than anger revealed 18 significant clusters, with the largest (2280 mm^3) located primarily in the left MFG (BA 9; see Figure 2 and Table 3). The ALE analysis of activation foci associated with anger greater than sadness revealed three significant clusters, with the largest (608 mm^3) located primarily in the right parahippocampal gyrus (BA 35; see Figure 2 and Table 3).

Sadness–Fear

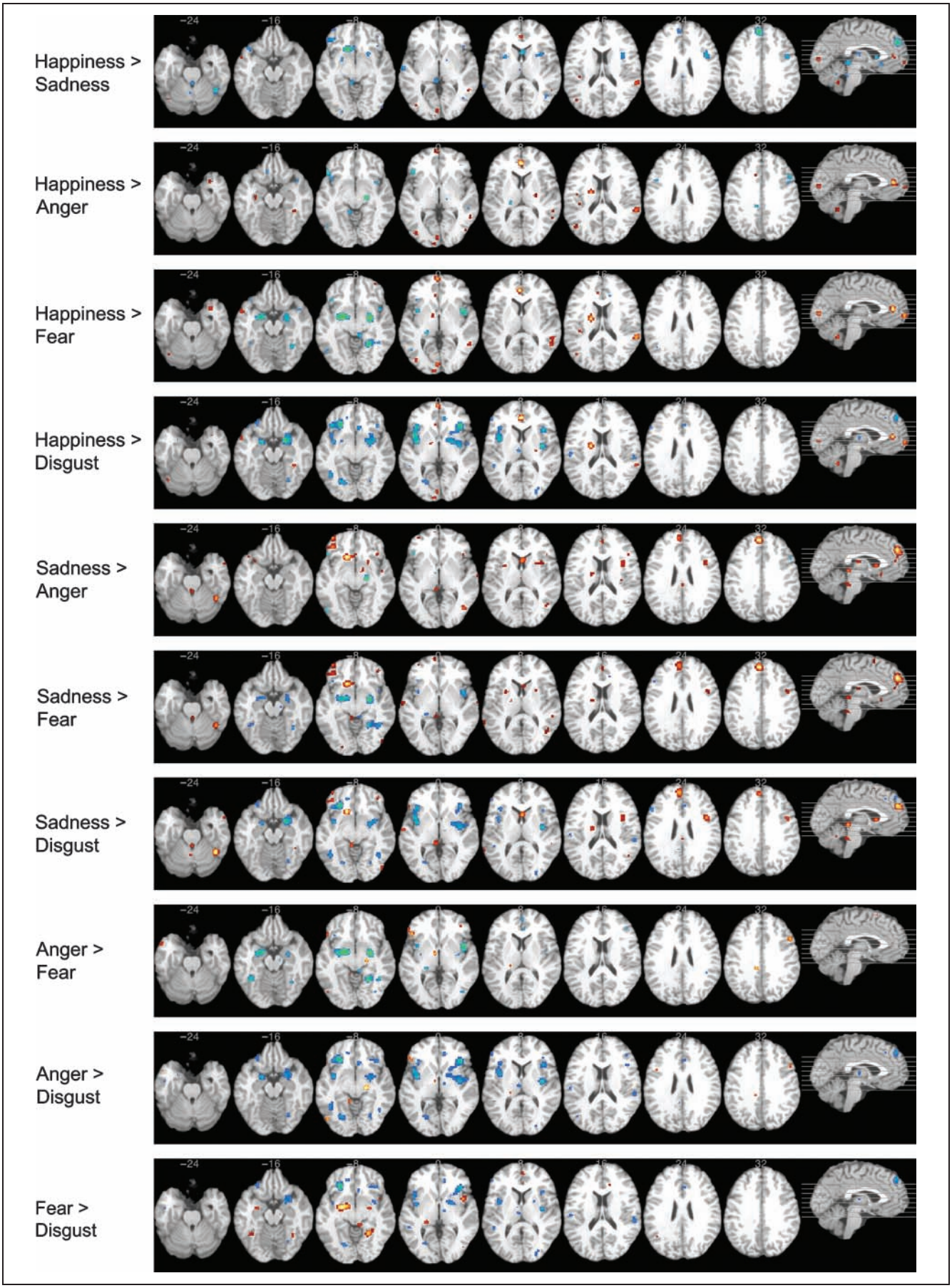
The ALE analysis of activation foci associated with sadness greater than fear revealed 14 significant clusters, with the largest (20840 mm^3) located primarily in the left medFG (see Figure 2 and Table 3). The ALE analysis of activation foci associated with fear revealed six significant clusters, with the largest (2632 mm^3) located primarily in the left amygdala (see Figure 2 and Table 3).

Sadness–Disgust

The ALE analysis of activation foci associated with sadness greater than disgust revealed 12 significant clusters, with the largest (1584 mm^3) located primarily in the right IFG (BA 9; see Figure 2 and Table 3). The ALE analysis of activation foci associated with disgust greater than sadness revealed 10 significant clusters, with the largest (6392 mm^3) located primarily in the left insula (see Figure 2 and Table 3).

Anger–Fear

The ALE analysis of activation foci associated with anger greater than fear revealed four significant clusters, with the largest (4784 mm^3) located primarily in the left IFG (BA 47; see Figure 2 and Table 3). The ALE analysis of activation foci associated with fear greater than anger revealed 11 significant clusters, with the largest (3688 mm^3) located primarily in the left putamen (see Figure 2 and Table 3).



Anger–Disgust

The ALE analysis of activation foci associated with anger greater than disgust revealed four significant clusters, with the largest (544 mm^3) located primarily in the left IFG (BA 47; see Figure 2 and Table 3). The ALE analysis of activation foci associated with disgust greater than anger revealed 15 significant clusters, with the largest (10696 mm^3) located primarily in the right putamen (see Figure 2 and Table 3).

Fear–Disgust

The ALE analysis of activation foci associated with fear greater than disgust revealed nine significant clusters, with the largest (2264 mm^3) located primarily in the left amygdala (see Figure 2 and Table 3). The ALE analysis of activation foci associated with disgust greater than fear revealed 12 significant clusters, with the largest (2328 mm^3) located primarily in the right putamen (see Figure 2 and Table 3).

Comparison with Previous Meta-analyses

The current meta-analysis identified consistent and discriminable patterns of neural activation associated with each basic emotion state. To further investigate the differences between the current findings and the findings of previous meta-analyses, we examined whether these differences were the result of the inclusion of additional data and/or the use of a more sensitive meta-analytic method (ALE). Specifically, we compared our findings to those that would have been obtained were we to limit our data only to the studies included the previous meta-analyses. That is, we kept analysis method constant and varied the specific studies included to match the studies examined by Murphy et al. (2003). Phan et al. (2002) did not directly address the differentiability of emotion states in their analyses, and thus it was not necessary to reanalyze their data separately from that of Murphy et al. (2003).

Murphy et al. (2003) did not find that the neural correlates of happiness and sadness could be differentiated based on the distribution of activations across the eight spatial divisions of the brain they analyzed. In contrast, we found that the ALE method was able to discriminate between these two emotions, in addition to all pairwise emotion comparisons (see Table 4), using the same data set used by Murphy et al. Furthermore, the areas that differentiated basic emotion states when ALE was applied to the prior data set substantially overlapped corresponding regions in the current meta-analysis. For example, 7 of 10 pairwise contrasts between emotion states using the data set of Murphy et al. revealed clusters that matched at least one of the three largest clusters for the corresponding pairwise contrasts in the current meta-analysis.

In summary, we were able to differentiate between each of the basic emotions with the smaller data set, even in cases where this was not possible in the original study, which used a different meta-analysis method. In addition, there was notable overlap between the results of the ALE analysis using the Murphy et al. data set and the results of the current ALE meta-analysis. These results suggest that the greater sensitivity of the ALE method contributed an increased ability to discriminate between emotion states in the current meta-analysis. Furthermore, comparison of the results obtained with both data sets confirmed that the substantially larger number of studies we examined relative to previous studies also contributed significantly

Figure 2. Activation likelihood maps for pairwise emotion contrasts, representing regional activations discriminating between basic emotion states. Statistical maps of significant ALE clusters associated with all pairwise contrasts among emotion states. Clusters displayed in the red-yellow color gradient correspond to the emotion state that is being subtracted from in the contrast (i.e., the minuend; e.g., happiness); clusters displayed in the blue-green color gradient correspond to the emotion state that is being subtracted (i.e., the subtrahend; e.g., sadness). In the Happiness–Sadness contrast, the most prominent clusters associated with happiness are located in right STG (BA 22) and left ACC (BA 32). In the Happiness–Sadness contrast, the most prominent clusters associated with sadness are located in right MTG (BA 37) and left medFG (BA 9). In the Happiness–Anger contrast, the most prominent clusters associated with happiness are located in left ACC (BA 32) and right STG (BA 22). In the Happiness–Anger contrast, the most prominent clusters associated with anger are located in left IFG (BA 47) and right parahippocampal gyrus (BA 35). In the Happiness–Fear contrast, the most prominent clusters associated with happiness are located in right STG (BA 22) and left ACC (BA 32). In the Happiness–Fear contrast, the most prominent clusters associated with fear are located in bilateral amygdala. In the Happiness–Disgust contrast, the most prominent clusters associated with happiness are located in left ACC (BA 24) and left medFG (BA 10). In the Happiness–Disgust contrast, the most prominent clusters associated with disgust are located in bilateral amygdala. In the Sadness–Anger contrast, the most prominent clusters associated with sadness are located in left MFG (BA 9) and right insula (BA 13). In the Sadness–Anger contrast, the most prominent clusters associated with anger are located in the right parahippocampal gyrus (BA 35) and left IFG (BA 47). In the Sadness–Fear contrast, the most prominent clusters associated with sadness are located in left medFG (BA 9) and left caudate head. In the Sadness–Fear contrast, the most prominent clusters associated with fear are located in bilateral amygdala. In the Sadness–Disgust contrast, the most prominent clusters associated with sadness are located in right IFG (BA 9) and left MFG (BA 9). In the Sadness–Disgust contrast, the most prominent clusters associated with disgust are located in bilateral insula and right STG (BA 22). In the anger–fear contrast, the most prominent clusters associated with anger are located in left IFG (BA 47) and right MFG (BA 9). In the Anger–Fear contrast, the most prominent clusters associated with fear are located in left putamen and right insula (BA 13). In the Anger–Disgust contrast, the most prominent clusters associated with anger are located in left IFG (BA 47) and left fusiform gyrus (BA 19). In the Anger–Disgust contrast, the most prominent clusters associated with disgust are located in right putamen and left insula (BA 13). In the Fear–Disgust contrast, the most prominent clusters associated with fear are located in left amygdala and right parahippocampal gyrus (BA 19). In the Fear–Disgust contrast, the most prominent clusters associated with disgust are located in left putamen and right IFG (BA 47).

Table 3. ALE Activation Clusters Differentiating Each Basic Emotion State

Activation Focus				
x	*y*	*z*	*Region (>100 mm³)*	*Size*
Happiness–Sadness				
Happiness > Sadness				
59.7	−40.5	15.7	R STG[a]	424
−0.4	39.3	6.7	L ACC (BA 32)[a]	344
−36.7	−30.5	18.2	L insula (BA 13)	120
−0.7	57.2	−3.2	L medFG (BA 10)[a]	112
Sadness > Happiness				
43.4	−64.6	6.8	R MTG	2536
−4.2	46.9	30.5	L medFG (BA 9)[a]	1976
−10.7	17.2	−8.9	L caudate head[a]	1760
−1.7	−20.9	10.7	L thalamus[a]	888
−63.5	−47.4	7.0	L MTG	800
−21.1	−0.6	−7.8	L basal ganglia	624
40.6	21.4	−3.6	R IFG (BA 47)	528
43.8	21.1	12.5	R IFG (BA 45)	464
−37.5	14.7	−13.6	L IFG (BA 47)	464
39.8	6.40	21.7	R basal ganglia (Put)	408
−26.8	3.20	9.0	L basal ganglia (Put)	272
22.9	8.30	−6.8	R basal ganglia (Put)	272
Happiness–Disgust				
Happiness > Disgust				
0.1	38	8.1	L ACC (BA 24)	672
−18.5	−9.5	17.1	L thalamus	624
−0.9	58.4	−1.3	L medFG (BA 10)[a]	456
−13.4	−6.1	1.9	L basal ganglia (GP)	136
Disgust > Happiness				
30.5	4.9	−3.7	R basal ganglia (Put)[a]	12008
−34.7	14.7	−3.2	L IFG (BA 47/Insula)[a]	9040
−22.2	−70.9	−6.2	L lingual gyrus[a]	1680
−20.1	−2.6	−14.7	L amygdala	1184
−1.3	43.5	39.7	L medFG (BA 8)	904
−41.2	−58.7	−7.2	L fusiform gyrus[a]	520
26.9	−82.6	9.8	R cuneus	512
6.9	20.7	−8.8	R ACC (BA 32)	296
10.4	36.9	−0.3	R ACC	224
−49.3	36.3	9.5	L IFG (BA 46)	168
4.5	25.8	24.5	R ACC (BA 24)	120

Table 3. (*continued*)

Activation Focus				
x	*y*	*z*	*Region (>100 mm³)*	*Size*
Sadness–Anger				
Sadness > Anger				
−3.6	46.5	27.7	L MFG (BA 9)	2088
36.9	7.1	17.3	R insula (BA 13)	1528
−11.5	16.8	−8.9	Left insula[a]	1328
2.3	11.3	6.2	R caudate head[a]	912
43.6	−67.1	4.0	R ITG	784
−37.8	35.3	−9.0	L MFG (BA 11)	768
−35.6	49.2	−4.0	L MFG (BA 10)	736
41.4	−51.7	−23.6	R cerebellum[a]	608
−3.5	−37.0	−3.1	L cerebellum[a]	400
−16.5	−11.4	12.5	L thalamus[a]	400
44.1	21.5	12.5	R IFG (BA 45)	328
39.3	21.7	−4.4	R IFG (BA 47)	328
12.9	−4.1	−6.1	R basal ganglia (GP)	256
−0.7	−19.5	10.9	L thalamus[a]	216
−32.4	11.7	−13.6	L IFG (BA 13)	192
22.7	8.4	−6.1	R basal ganglia (Put)	176
−6.6	59.9	2.7	L medFG (BA 10)	152
32.9	−22.0	18.8	R insula (BA 13)	120
Anger > Sadness				
19.9	−18.8	−9.0	R PHG	2536
−43.8	22.4	−4.1	L IFG (BA 47)[a]	1976
−46.5	−74.0	−10.7	L fusiform gyrus[a]	1760
Anger–Fear				
Anger > Fear				
−47.1	25.2	−2.9	L IFG (BA 47)[a]	784
48.6	13.9	30.1	R MFG (BA 9)	520
−7.9	−34.2	31.9	L cingulate gyrus[a]	176
20.2	−20.8	−7.9	R PHG	152
Fear > Anger				
−21.4	−6.9	−10.6	L basal ganglia (Put)[a]	3688
33.8	−4.3	−4.7	R insula (BA 13)[a]	3512
28.7	−52.6	−7.9	R cerebellum[a]	2080
−37.5	−52.5	−16.0	L fusiform gyrus[a]	920
3.4	47.9	5.2	R ACC (BA 32)[a]	440
42.5	−40.2	20.5	R insula (BA 13)[a]	304

Table 3. (*continued*)

Activation Focus				
x	*y*	*z*	*Region (>100 mm³)*	*Size*
37.5	10.3	19.6	R insula (BA 13)[a]	296
−35.8	4.3	1.5	L insula	248
41.8	33.6	15.3	R MFG (BA 46)	224
−21.1	−34.1	−0.7	L PHG[a]	208
4.8	34.0	6.0	R ACC (BA 24)	144
Anger–Disgust				
Anger > Disgust				
−46.0	25.9	−2.9	L IFG (BA 47)[a]	544
−45.6	−74.2	−11.2	L fusiform gyrus[a]	480
19.4	−20.9	−8.4	R PHG (BA 35)	456
49.1	16.2	30.6	R MFG (BA 9)	112
Disgust > Anger				
32.0	5.3	−2.2	R basal ganglia (Put)[a]	10696
−33.5	14.2	−3.2	L insula (BA 13)	7624
−22.6	−70.8	−6.0	L lingual gyrus[a]	1456
−19.5	−2.9	−15.5	L PHG	1008
−1.3	43.4	40.1	L medFG (BA 8)	936
−40.9	−52.7	−8.4	L fusiform gyrus[a]	648
6.9	20.7	−8.8	R ACC (BA 32)	280
10.4	36.5	−0.2	R ACC	240
41.9	−60.3	−6.24	R fusiform gyrus	232
−18.5	−50.5	−3.49	L PHG (BA 19)	200
−45.9	−10.1	−20.77	L temporal lobe (BA 20)	176
−49.2	36.3	9.38	L IFG (BA 46)	152
4.4	25.7	24.46	R ACC (BA 24)	144
41.6	34.9	16.13	R MFG (BA 46)[a]	128
−12.0	38.0	−7.01	L medFG (BA 10)	128
Happiness–Anger				
Happiness > Anger				
−0.5	39.5	8.3	L ACC (BA 32)[a]	1032
58.4	−40.6	14.4	R STG (BA 22)[a]	824
−4.2	−91.8	2.2	L lingual gyrus	576
−18.2	−9.8	17.0	L thalamus	496
−3.2	59.5	0.2	L medFG (BA 10)[a]	200
−35	−31.5	18.0	L insula (BA 13)	128
Anger > Happiness				
−43.4	21.4	−4.5	L IFG (BA 47)[a]	1536

Table 3. (*continued*)

Activation Focus				
x	*y*	*z*	*Region (>100 mm³)*	*Size*
19.6	−19.8	−8.6	R PHG	808
48.0	13.2	30.2	R IFG (BA 9)	752
36.4	6.3	−10.7	R IFG (BA 13)[a]	344
−43.9	10.6	26.3	L IFG (BA 9)[a]	336
−11.0	24.0	−16.0	L medFG (BA 25)	112
Happiness–Fear				
Happiness > Fear				
55.3	−45.7	9.8	R STG (BA 22)[a]	1592
−2.7	38.3	9.8	L ACC (BA 32)[a]	776
−18.3	−8.7	16.7	L thalamus	672
−1.9	58.6	−1.9	L medFG (BA 10)[a]	592
−5.3	31.6	−2.8	R ACC (BA 32)	192
−36.7	−31.4	17.9	L insula (BA 13)	144
Fear > Happiness				
−21.1	−6.2	−10.8	L amygdala[a]	3192
24.6	−8.3	−11.3	R amygdala[a]	2600
27.8	−52.7	−9.3	R fusiform gyrus[a]	2072
42.9	2.7	−1.8	R STG[a]	2056
−38.1	22.1	−7.7	L IFG (BA 47)	896
−46.1	−63.3	−4.2	L middle Occ gyrus[a]	568
−35.3	5.0	1.0	L insula	424
38.2	10.2	19.9	R insula (BA 13)[a]	288
42.8	−40.7	20.4	R insula (BA 13)[a]	192
13.3	29.5	13.9	R ACC (BA 32)[a]	168
5.3	47.9	4.6	R medFG (BA 10)	120
Sadness–Fear				
Sadness > Fear				
−3.7	47.0	27.5	L medFG (BA 9)[a]	2840
−11.6	17.5	−8.5	L caudate head[a]	1248
44.2	5.5	28.1	R IFG (BA 9)	816
−39.0	35.2	−8.8	L cerebellum[a]	752
41.1	−51.7	−22.6	R MFG (BA 10)	704
2.3	7.8	61.7	R precentral gyrus	592
−36.7	49.2	−5.7	R cerebellum[a]	560
−4.4	−38.4	−4.8	R thalamus	552
−16.5	−10.5	12.2	R MFG (BA 11)	552

Table 3. (*continued*)

Activation Focus				
x	*y*	*z*	*Region (>100 mm³)*	*Size*
44.2	−62.9	8.4	R cerebellum[a]	464
−47.4	−6.7	41.1	L caudate head[a]	456
−60.0	−14.7	−0.7	L MFG (BA 47)[a]	312
39.4	39.0	−10.7	R medFG (BA 10)	280
−0.6	−19.9	10.9	L basal ganglia (Put)	112
Fear > Sadness				
−20.4	−7.1	−10.2	L PHG/amygdala[a]	2632
24.0	−10.3	−10.4	R midbrain	2504
32.2	−52.9	−8.6	R fusiform gyrus[a]	2328
42.5	1.1	0.2	R insula (BA 13)[a]	1376
4.3	47.4	3.6	R ACC (BA 32)[a]	336
−38.4	−52.1	−17.9	L IFG (BA 47)	304
Sadness–Disgust				
Sadness > Disgust				
41.1	5.8	24.1	R IFG (BA 9)	1584
−4.1	48.1	26.9	L medFG (BA 9)[a]	1520
40.4	−51.3	−22.6	R cerebellum[a]	1024
−4.5	−38.7	−4.6	L cerebellum[a]	808
−13.0	16.2	−9.1	L insula[a]	800
1.4	11.0	6.2	R caudate head[a]	664
−16.5	−10.7	14.0	L thalamus	536
2.3	7.7	61.6	R MFG (BA 47)[a]	456
−47.3	−6.9	41.0	L precentral gyrus	440
−40.3	35.7	−8.4	L MFG (BA 47)	376
−37.7	49.5	−6.6	R MFG (BA 11)	176
−29.3	49.3	4.0	L MFG (BA 10)	128
Disgust > Sadness				
−33.7	15.3	−3.6	L IFG (BA 47)	6392
30.5	−3.8	−5.8	R STG (BA 22)[a]	6288
35.6	23.3	0.9	R insula (BA 13)[a]	1144
−22.8	−69.8	−4.3	L lingual gyrus[a]	600
−49.9	19.1	26.3	L IFG (BA 9)	560
−41.7	−56.2	−7.8	L fusiform gyrus[a]	448
−19.5	−2.8	−16.6	L PHG/amygdala	432
40.4	−57.7	−8.6	R fusiform gyrus	424
−13.0	38.3	−7.5	L medFG (BA 10)	136
−2.5	43.7	42.5	L medFG (BA 8)	112

Table 3. (*continued*)

Activation Focus				
x	*y*	*z*	*Region (>100 mm³)*	*Size*
Fear–Disgust				
Fear > Disgust				
−20.6	−8.5	−9.7	L amygdala[a]	2264
24.5	−51.5	−7.6	R PHG (BA 19)[a]	992
42.6	6.4	−2.1	R insula (BA 13)[a]	600
−38.8	−54.7	−16.0	L cerebellum	432
4.1	48.3	5.2	R ACC (BA 32)[a]	352
25.0	−11.0	−10.4	R amygdala[a]	328
−20.8	−33.7	−0.3	L PHG (BA 27)[a]	256
42.2	−39.8	20.1	R insula (BA 13)[a]	208
13.3	29.0	13.8	R ACC (BA 32)[a]	112
Disgust > Fear				
34.2	22.7	−0.9	R basal ganglia[a]	2328
−25.6	27.8	−10.0	L IFG (BA 47)	2192
−38.7	3.6	0.9	L insula (BA 13)	2088
26.5	4.4	−14.6	R IFG (BA 47)[a]	1792
28.1	−5.2	3.5	R basal ganglia[a]	1544
−1.5	43.5	39.9	L medFG (BA 8)	888
−19.6	−71.4	−6.1	L lingual gyrus[a]	736
27.0	−82.3	10.1	R cuneus (BA 30)	448
−47.3	−43.7	3.9	L MTG (BA 22)	432
−13.0	38.2	−6.7	L medFG (BA 10)	256
10.6	36.6	−0.9	R ACC	136
4.1	25.5	24.7	R ACC (BA 24)	120

Labels (e.g., "Happiness > Sadness") indicate regions of consistently greater activity (i.e., activation likelihood) for the first emotion relative to the second. Each cluster greater than 400 mm³ in size is reported, along with the weighted central activation likelihood focus, the region corresponding to the cluster with the highest ALE score within the cluster, and the total cluster size in mm³. Additional clusters of interest that surpassed a threshold of 100 mm³ were also reported. L and R indicate ALE clusters located in the left and right hemispheres, respectively. Inf = inferior; Occ = occipital; GP = globus pallidus; Put = putamen; PHG = parahippocampal gyrus. BA labels are provided to differentiate ALE clusters in larger regions that occur in multiple contrasts.

[a]Indicates regions that overlapped with the reanalysis that involved only studies that used facial expressions.

to the analysis, by allowing additional ALE clusters to be identified that discriminated between basic emotions.

Role of Stimulus Differences

The studies contributing to the activation foci in the ALE analysis used a wide range of experimental materials and methods to examine emotion, such as facial expressions

Table 4. ALE Activation Clusters Differentiating Each Basic Emotion State for Reanalysis with Reduced Data Set

Contrast	*Regions*
Happiness > Sadness	L ACC [BA 32] (1264 mm^3), R MTG, L MTG, L insula, R STG
Sadness > Happiness	R ACC [BA 24] (2096 mm^3), L caudate head, R insula, L medFG, L cerebellum, L SFG, L MFG, R insula, R MFG, L thalamus, R medFG
Happiness > Anger	L ACC [BA 32] (1216 mm^3), L cerebellum, R MTG, L MTG, R Put, L insula, L thalamus
Anger > Happiness	R IFG (1552 mm^3), R thalamus, L STG, L cingulate gyrus, R PHG, L IFG, L thalamus, L cerebellum, R cingulate gyrus, R MFG, L MFG
Happiness > Fear	L ACC [BA 24] (1240 mm^3), R MTG, L medFG, R STG, R posterior cingulate, L insula, R ACC [BA 32]
Fear > Happiness	L amygdala (3504 mm^3), R insula, R Put, R thalamus, R cingulate gyrus, L SFG, L IFG, R PHG, L thalamus
Happiness > Disgust	L ACC [BA 24] (2528 mm^3), L medFG, L cerebellum, R MTG, L MTG, R STG, R supramarginal gyrus, L GP, L ACC [BA 32], L thalamus, L insula, R Put
Disgust > Happiness	L insula (3024 mm^3), R STG, R Put, R postcentral gyrus, R cuneus, L thalamus, R IFG (Insula)
Sadness > Anger	L MFG (1068 mm^3), R MFG, R caudate head, R insula, L medFG, L thalamus, R IFG, L MTG
Anger > Sadness	L IFG, (2256 mm^3), R cingulate, L fusiform gyrus, R PHG
Sadness > Fear	L caudate head (912 mm^3), R MFG, R IFG, R thalamus, R cerebellum, L Put
Fear > Sadness	L amygdala (2734 m^3), R insula, R fusiform gyrus, L IFG
Sadness > Disgust	L medFG (856 mm^3), R caudate head, L cerebellum, L thalamus, R MFG, L MFG, L medFG
Disgust > Sadness	R STG (5478 mm^3), L insula, L amygdala, R insula, L fusiform, R insula, R Put
Anger > Fear	L IFG (982 mm^3), L MFG, R MFG, L cingulate gyrus
Fear > Anger	R insula (4913 mm^3), L Put, L amygdala, R ACC [BA 32], L insula, L fusiform gyrus, L PGH, L thalamus
Anger > Disgust	L IFG (1092 mm^3), L STG, L fusiform gyrus, R PHG, L cerebellum, R ACC [BA 32], L cingulate gyrus, L thalamus, L MFG, R MFG, R cingulate gyrus, L Put, L medFG
Disgust > Anger	R STG (1608 mm^3), R GP, R postcentral gyrus, L thalamus, R IFG (Insula), L MTG
Fear > Disgust	L amygdala (4544 mm^3), R cingulate gyrus, L SFG, R insula, R precentral gyrus, L thalamus, R thalamus, R fusiform gyrus, L IFG, R STG, R PHG, R Put, R thalamus, R ACC [BA 32]
Disgust > Fear	R Put (2200 mm^3), L GP, R postcentral gyrus, L insula

Each cluster greater than 400 mm^3 is reported. The region corresponding to the largest cluster is reported first, with the total cluster size listed in parentheses. Additional clusters of interest that surpassed a threshold of 100 mm^3 are also reported. L and R indicate ALE clusters located in the left and right hemispheres, respectively. Inf = inferior; GP = globus pallidus; Put = putamen; PGH = parahippocampal gyrus. BAs are provided to differentiate activations in larger regions that occur in multiple contrasts.

of emotion, emotional pictures, films, and scripts. Because studies differed in the frequency with which they used specific types of stimuli and elicitation methods, we examined whether such methodological differences could have contributed to the neural differences observed here. Notably, facial expressions of emotion were the most frequently used stimulus type for studies examining all basic emotions except for disgust, where emotional pictures were the second most frequent stimulus type. Specifically, facial expressions were used as stimuli in 14 of 30 happiness studies, 11 of 33 sadness studies, 10 of 16 anger studies, 24 of 37 fear studies, and 9 of 29 disgust studies (11 of 29 disgust studies used picture stimuli). Because of insufficient numbers of associated studies, it was not possible to examine the differential effects of every type of stimulus. Accordingly, we focused on the potential role of the most commonly used stimulus type, facial expressions.

To investigate the potential effects of stimulus material on the activation patterns associated with a given emotion, we conducted the ALE analysis a second time, including only those studies that used facial expressions as stimuli. In this way, we ruled out the possibility that systematic differences in stimulus type could contribute to activation differences differentiating basic emotions. Based on the hypothesis that stimulus differences did not contribute significantly to our original ALE results, we expected to obtain roughly similar results when we controlled for stimulus differences in this manner, although we also expected that the results would differ somewhat because of the smaller number of studies. The results of this reanalysis confirmed that the ALE results obtained with studies using facial emotion stimuli were similar to the results of the original analyses for each basic emotion. Overall, there was substantial overlap in the number of regional clusters

identified in both analyses (Table 2). Furthermore, the regions that were central to the differentiation of each basic emotion state in the original analyses were also typically significant in the analysis limited to studies using facial emotion stimuli (Table 3). These results suggest that differences in stimulus type did not drive the primary finding of significant differentiation of emotion states because when the potential effects of stimulus differences were eliminated, the characteristic patterns of neural activation associated with each basic emotion were still observed, and each basic emotion could still be differentiated on the basis of regional activations.

DISCUSSION

The primary goal of this study was to assess the extent to which the current neuroimaging literature supports the proposal of basic emotion theories that different basic emotion states are associated with consistent, characteristic, and discriminable patterns of brain activity. The results of the ALE meta-analysis supported the predictions of basic emotion theories. Each of the basic emotion states examined (anger, fear, sadness, anger, and disgust) was consistently associated across studies with characteristic patterns of regional brain activity. For example, across a variety of different experimental paradigms and stimuli, we found that fear was associated with increased activation in the amygdala and insula, relative to emotionally neutral stimuli. Importantly, each basic emotion was reliably distinguished or differentiated from the other emotions on the basis of its characteristic pattern of brain activation. Specifically, every pairwise statistical contrast between the activation foci associated with emotion states (e.g., fear vs. anger) in the ALE analysis yielded a set of regional brain activations that reliably differentiated between each pair of emotions. Further, as predicted, the signature patterns of neural activation that characterized each emotion also most consistently differentiated that emotion from other emotions. This is in contrast with other possible scenarios, for example, where the regions that differentiate between emotions could have little overlap with the core, characteristic brain regions consistently activated by each emotion. Finally, the associations between emotion states and regions of brain activation identified in our ALE meta-analysis of the neuroimaging literature converge with the findings from other approaches including neuropsychological studies (e.g., Adolphs, et al., 1994) and studies of nonhuman animals (e.g., Davis, 1992, 1994).

The current meta-analysis found that all five basic emotion states were associated with consistent and discriminable patterns of neural activation (Figure 2). Happiness consistently activated rostral ACC and right STG, and activity in both regions differentiated happiness from sadness, anger, fear, and disgust (ACC only). Sadness consistently activated MFG and head of the caudate/subgenual ACC, and activity in both regions reliably differentiated sadness from happiness, anger, fear, and disgust. Anger consistently activated IFG and PHG, and both regions differentiated anger from all other emotion states. Fear consistently activated amygdala and insula, and these regions differentiated fear from happiness, sadness, anger (insula only), and disgust (posterior insula). Disgust consistently activated IFG/anterior insula, and these regions reliably differentiated disgust from all other emotion states. Together, these findings support the predictions of basic emotion theories by demonstrating that basic emotion states are associated with consistent patterns of brain activation and that these patterns differ significantly between emotions.

In contrast to the current meta-analysis, two previous meta-analyses (e.g., Murphy et al., 2003; Phan et al., 2002) found more limited support for basic emotion theories. Phan et al. (2002), using a meta-analytic method based on counts of activated regions, found limited evidence for consistent associations between brain regions and basic emotions. For example, fear was more consistently associated with amygdala activation than any other emotion state, and sadness exhibited a greater association with subcallosal cingulate cortex activation in comparison to other emotions. Anger, happiness, and disgust did not consistently activate any brain region more than other emotions states. However, Phan et al. did not directly contrast activation patterns associated with each basic emotion, so the extent to which these activations composed patterns that discriminated between basic emotions could not be addressed. Murphy et al. (2003) did address this question and found reliably different spatial patterns of activation neural correlates for fear (amygdala), disgust (insula), and anger (globus pallidus and lateral OFC). However, happiness and sadness were not reliably differentiated, and the spatial divisions used in that study were too large to address the issue of discriminability at the level of specific brain regions.

Our meta-analysis differed from these previous meta-analyses in two important ways. We included a substantial amount of new data from thirty studies that were not included in the largest meta-analysis to date, and we used the more spatially sensitive ALE method. To determine the extent to which our method (ALE) versus the inclusion of more data contributed to the increased ability to differentiate between neural patterns associated with basic emotions, we used the ALE method to analyze the smaller data set analyzed by Murphy et al. (2003) and compared the results to those of the current meta-analysis. The results demonstrated that the ALE method was able to differentiate between all of the emotion states, including the pair of emotions that the previous meta-analysis was not able to differentiate. These findings suggest that both the increased sensitivity of the ALE method and the inclusion of additional studies contributed to the increased ability to discriminate among emotions.

Converging evidence from several domains suggests that discrete basic emotions are psychologically, physiologically, and neurologically discriminable (e.g., Rainville, Bechara, Naqvi, & Damasio, 2006; Murphy et al., 2003; Ekman, Levenson, & Friesen, 1983). For example, therapeutic

intervention studies of depression have demonstrated that reduction in depressive symptoms is associated with increased activity in BA 24 (cingulate cortex), when deep brain stimulation or cognitive behavioral therapy is used (Mayberg et al., 2005; Goldapple et al., 2004), and decreased activity in BA 9 (medial frontal cortex), when cognitive behavioral therapy is used (Goldapple et al., 2004). Mood fluctuations associated with happiness versus sadness may be supported by subregions of BA 24 (e.g., subgenual ACC; Mayberg et al., 2005) that have subcortical projection to the brainstem and thalamus (areas that are involved in circadian rhythm maintenance; Barbas, Saha, Rempel-Clower, & Ghashghaei, 2003; Ongur, An, & Price, 1998). These findings correspond with our results that implicate ACC (BA 24) and medFG (BA 9) are uniquely associated with happiness and sadness, respectively. Similarly, our results suggest an important role for IFG in anger, and this finding is complemented by the results of neuropsychological studies which indicate that damage to the IFG can increase violent and aggressive behaviors, consistent with a proposed regulatory role for the IFG in the expression of anger (Grafman et al., 1996; Damasio, Grabowski, Frank, Galaburda, & Damasio, 1994). The IFG may be engaged during exposure to angering stimuli as an automatic control to curb the potential for an overreaction such as unbridled rage. In addition, we found that disgust was associated with activity in the insula, and stimulation of this region has been shown to induce nausea (Penfield & Faulk, 1955) and unpleasant sensations in the throat mouth and nose (Krolak-Salmon et al., 2003); both of which are involved in the experience of disgust. The visceral feeling that people experience in response to a disgusting stimulus may therefore reflect automatic simulation of these sensations, supported by the insula. Finally, the current meta-analytic review confirmed an important functional role for the amygdala in fear. The relationship between amygdala and fear is perhaps the most robust structure–function association found across studies, with converging evidence from meta-analyses of neuroimaging studies (e.g., Murphy et al., 2003; Phan et al., 2002), animal models of fear (Davis, 1994), single-unit recording studies (Maren, 2001), and human lesion studies (Adolphs et al., 1994). The amygdala has been shown to direct attention to threat cues by modulating activity in primary visual cortex, as evidenced by effective connectivity (Pessoa, McKenna, Gutierrez, & Ungerleider, 2002) and lesion research (Vuilleumier, Richardson, Armony, Driver, & Dolan, 2004). In addition, it has been suggested that amygdala activity may also indirectly influence thought and behavior through the modulation of prefrontal activity (Miller & Cohen, 2001), although this claim requires further exploration. A fearful response to a threatening stimulus may recruit the amygdala to focus attention to relevant cues and initiate an appropriate response to the threat.

Although our goal was to investigate the neural activations associated with basic emotions across a variety of contexts and elicitation methods, it is important to note that certain stimulus types were represented more than others in the studies comprising our meta-analysis. For example, facial emotion stimuli were the most frequently used type of stimulus in studies of happiness, sadness, anger, and fear. To examine the potential influence of stimulus differences on the results of our meta-analysis, we conducted an additional ALE analysis limited to studies that used facial expressions as stimuli. The results demonstrated that all five basic emotions were associated with unique and reliable patterns of neural activation, even when the analysis was limited to one stimulus type. Furthermore, the regions identified by this analysis overlapped with the regions identified by the original consistency and discriminability analyses. These findings suggest that the primary finding, that the ALE analysis could differentiate between basic emotions on the basis of neuroimaging evidence, was not driven by stimulus material differences. Because the majority of neuroimaging studies used facial expression stimuli, a remaining issue is the extent to which these findings generalize to other emotional stimuli. As a first step toward addressing this issue, we examined all the studies that did not use facial emotion stimuli, in an ALE analysis, and observed a broadly similar pattern of regions differentiating basic emotions. These results provide some preliminary evidence to suggest that our primary ALE results are not unique to studies using facial emotion, but these results should be viewed as only preliminary because of both the substantially smaller data set (limiting the applicability of the ALE method) and variation across basic emotions in the number of studies that used stimulus types. As additional neuroimaging studies continue to adopt a wider range of stimuli, future meta-analyses will be able to better address this issue.

Regarding limitations of this study, the spatial sensitivity of the current meta-analysis was limited by the resolution of the neuroimaging data in the studies analyzed (approximately 64 cubic mm voxels for fMRI). Subsequent data processing steps and summarization for publication further reduced the effective spatial resolution in individual studies. Another potential source of bias was the fact that a small minority of studies (12% of foci from all studies) gave preference in their analyses to a priori ROIs by using more lenient thresholds for these regions, which would tend to increase the representation of these regions in the ALE analysis. Notably, the majority (72%) of these studies examined the neural correlates of fear and disgust, and thus any potential bias would be primarily limited to these two basic emotions. We examined the effect of excluding these foci obtained with more lenient thresholds from the ALE analyses and found that their exclusion resulted in minimal and nonsignificant changes in the outcome of the meta-analysis.

The ALE method also makes some simplifying assumptions that may affect the relative influence of individual activations and individual studies. All activation maxima above the significance threshold adopted in a particular study are given equivalent weight in the analysis, so that

variations in activation intensity are not accounted for. Similarly, studies with greater numbers of activation maxima will contribute more to the ALE map than studies with fewer maxima, although inspection of our individual studies did not reveal any systematic relationship between the number of maxima per study and the results of the consistency and discriminability analyses. In addition to these considerations, the requirements of the analysis (e.g., analyses of whole-brain data) necessarily limited the number of studies that were included in the review. Another potential limitation includes publication biases such as the file-drawer problem (tendency for null findings not to be published), which is unavoidable.

The ALE approach taken here assessed correspondences between emotional processing and individual brain regions rather than networks of regions. However, interactions between brain regions have been demonstrated to contribute importantly to emotion processing, and thus future meta-analyses should examine interactions and functional networks. Furthermore, we cannot conclude that these results reflect brain regions associated with the induction of basic emotion states because, like all previous meta-analytic studies, we included studies that addressed a wide range of emotion-related processes so that we could investigate the core neural signatures associated with basic emotions across a variety of contexts. As the neuroimaging literature progressively incorporates a wider range of stimuli and methods exploring the neural correlates of basic emotions, this will facilitate the characterization of the effects of induction method and stimulus material.

Although we focused on differentiating basic emotions on the basis of brain activation patterns, a recent meta-analysis used a complementary approach and a different voxel-based meta-analytic method (multilevel kernel density analysis) to explore the functional grouping of emotion-related activations in the brain (Kober et al., 2008). This study used a data-driven approach that ignored emotion labels such as happiness and sadness. Instead, Kober et al. (2008) investigated the multivariate patterns of coactivation that emerged when activations from neuroimaging studies of emotion are examined, identifying six functionally distributed networks. Because Kober et al. (2008) explicitly avoided analyzing activations on the basis of basic emotion categories, it is difficult to compare between their results and those of the current study. The current meta-analysis also did not examine contextual, linguistic, and other influences on emotion states and their neurobiological correlates. We acknowledge that the experience and interpretation of emotional states can be strongly influenced by situational factors, both internal and external, and thus brain activity would be expected to reflect these factors. However, we sought to investigate the reliability of neural patterns associated with basic emotion categories and thus did not explore the factors contributing to their variability here.

Emotions have been characterized by both dimensional and categorical theoretical frameworks. Dimensional views of emotion have proposed that emotions can be characterized in terms of component dimensions such as arousal (emotional strength) and valence (pleasantness vs. unpleasantness). The dimensional approach to emotion has proven highly successful in accounting for a wide range of emotional phenomena and is theoretically more parsimonious than categorical approaches such as basic emotion theories (Lang, Bradley, & Cuthbert, 1990; Watson & Tellegen, 1985). Although dimensional and basic emotion theories have sometimes been characterized as being incompatible in some respects (e.g., Barrett, 2006), they are not necessarily mutually exclusive characterizations of emotional experience. A hybrid view combining dimensional descriptions of emotion states in terms of arousal and valence with additional characterization provided by basic emotion categories would be consistent with the current findings. For example, whereas a dimensional description in terms of arousal and valence can concisely characterize key aspects of emotional reactions to a photograph eliciting disgust, the basic emotion categorization of disgust captures facets of the experience of disgust not conveyed by the dimensional description, such as a somatic state of nauseation, elicitation of a facial expression of disgust, and CNS activation of the consistent and discriminable regional brain activations identified in the current study. Regarding the neural substrates corresponding to affective dimensions, several neuroimaging studies have identified discriminable neural correlates of emotional arousal (e.g., amygdala) and valence (e.g., subregions of pFC; Lewis, Critchley, Rotshtein, & Dolan, 2007; Dolcos, LaBar, & Cabeza, 2004; Anderson, Christoff, Panitz, De Rosa, & Gabrieli, 2003). Taken together, the results of these studies and the current meta-analysis results indicate that both dimensional views and basic emotion views are supported by neuroimaging studies in the sense that the constructs associated with each view have identifiable neural correlates as assessed with neuroimaging. Further research into the interplay between neural mechanisms underlying basic emotions and corresponding mechanisms associated with arousal and valence dimensions will help elucidate how each contributes to emotional experience and behavior.

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